Guest Article

Changing Definition and Current Treatment of Preeclampsia.

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Changing Definition. Because of unknown aetiology of preeclampsia, there is lack of uniformity of its constituent parts.

Old Definition. Towards the earlier part of this century name of preeclampsia was preeclamptic toxaemia (PET) in British practice. The disease was described as syndrome of a triad of hypertension at/above 140/90 mmHg, oedema and albuminuria developed during second half of pregnancy. Toxaemia was derived from placental toxin theory which was unproved. This terminology, preeclamptic toxaemia prevailed till midsixties. Thereafter preeclampsia was defined as any two of the three signs - thus combination of hypertension and oedema or hypertension and albuminuria or even oedema and albuminuria were taken as preeclampsia. From mid70s the definition was hypertension at and above 140/90 mmHg plus oedema or proteinuria or both. Herein hypertension became the key sign.

New Definition. International society for study or hypertension in pregnancy (ISSHP), 1986 defines preeclampsia as persistent hypertension of at least 140/90 mmHg after 20 weeks of gestation plus total protein excretion (proteinuria) of greater than 300 mg/24 hours. In this definition, two things are emphasised. (1.) Hypertension is induced by pregnancy (PIH) and becomes the basic sign of preeclampsia. (2.) Albuminuria is replaced by proteinuria as because there is excretion of both albumin and globulin. Thus PIH is not preeclampsia. Preeclampsia is PIH plus proteinuria.

Currently preeclampsia remains a syndrome of two signs - hypertension plus proteinuria. The term preeclamptic toxaemia is now obsolete.

Generalised oedema develops in 85% of preeclampsia and is considered the associated clinical sign of preeclampsia but not a sign of preeclampsia (Dawn, 1987). Redman, 1995 opines that oedema should not be included in definition of preeclampsia. In older definition too much emphasis was made on oedema even in absence of hypertension.

Clinical practice even today associates excess maternal weight gain with development of preeclampsia. Physiological oedema is found in 80% pregnancy and has not been shown to be precursor of pathological oedema (Robertson 1991). The author identified pathological generalised oedema associated with preeclampsia as a separate entity from physiological oedema and develops as a generalised oedema from the beginning during second half of pregnancy. Generalised oedema results from silent intake of excess of salt and drinking of excess water by the woman. Ten percent primigravida woman who develops preeclampsia has a nervous system with salt

and water crazy centres and also emotional. Some of them even in early 20s can not sleep well at night or in disturbed sleep.

Working on Dawn Hypothesis for preeclampsia (1987). The author could explain in his hypothesis for preeclampsia the cause of pregnancy induced hypertension as : (1.) Familial hypertensive diathesis (in 10% primigravidae). (2.) Gestational vasoconstrictors (angiotensin II). (3.) Dietary salt overloading (4.) Stress interaction. Preeclampsia prone women suffers from stress more. Four components of the hypothesis are explained further as :

1. Familial hypertensive diathesis in family tree can be identified in parents and kins of almost all women developing PIH by checking blood pressure of parents and kins (mostly in asymptomatic stage of hypertension). Chesley et al, 1968 also could identify familial hypertension as predisposing factor to eclampsia.

2. Uteroplacental complex liberates vasoconstrictors (angiotensin II). This causes hypertensive diathesised vascular tree of PIH prone woman to be vasoactive.

3. PIH developing women are identified as salt crazy loading her system with daily salt intake more than 10 gm. Sodium shifting in arteriolar wall causes vasoconstriction raising blood pressure to 140/90 mmHg (mild PIH).

4. Stress (physical and mental) aggravates hypertension if left uncared for in a dramatic way.

   Oedema is caused by salt and water overloading. Those who develop oedema have predisposition for it.

   Dry PIH (without oedema) is a chronic hypertension being aggravated by pregnancy.

Treatment.

Prevention. Low dose aspirin prophylaxis 50-80 mg daily with placebo control was tried in multiple centres since

Down staging of preeclampsia and its progression.

	Stage I (mild preeclampsia)	Stage II (Severe preeclampsia)	Stage III (Eclampsia)
Hypertension	≥140/90 mmHg-160/110 mmHg	≥160/110 mmHg	≥160/110 mmHg
Proteinuria	0	+	++
Symptoms	0	+	Convulsion
Antihypertensive drug	Nil	Used	Mandatory
Diuretics	Nil	Selective	Used
Anticonvulsants	No	Possibly	Mandatory
Timing of admission	Elective	today	Emergency
Delivery	After 38 wks	After 24-36 wks	on stability

Rise of systolic pressure is now taken as significant as diastolic pressure.

1978. To mention a few important ones are :
American study (multicentric, 3135 primigravidae) on 60 mg aspirin and placebo control daily during second half of pregnancy showed no prevention of preeclampsia (Sibai et al, 1993).

The collaborative low dose Aspirin study in pregnancy (CLASP, 1994) organised by British Medical Council was a multicentric study (213 centres in 16 countries worldwide from January 1988 - December, 1992). Aspirin was given during 12-32 weeks gestation on randomized placebo controlled trial in 9384 pregnant women. Use of low dose aspirin in pregnant woman was not associated with any statistically significant reduction in incidence of preeclampsia. Thus low dose aspirin prophylaxis is not recommended in clinical practice.

Downstaging of preeclampsia - (Halting at stage I but preventing stage II) Development of mild preeclampsia (stage I) cannot be prevented. However downstaging at stage I (mild preeclampsia) is possible preventing severe preeclampsia (stage II). Thus stage III (eclampsia) could be also prevented.

Maternal care on rest and sleep, avoidance of extra salt regime.

All pregnant women are cared by Dawn Rule of Ten (Dawn 1997) by food and rest from 10th week with a goal of achieving average weight gain of 10 kg throughout pregnancy. Preeclampsia-prone woman is identified at 10th week from family history of hypertension, salt crazy habit, emotional temperament. She often cannot sleep at night.

At checkup husband always accompanies the wife, mother and in-laws at times. They are instructed to supervise the patient to follow the regime. She is given food chart having 5-10 gm salt daily but no extra salt, and salty food throughout pregnancy, 2 hours afternoon rest on lateral lying and having good sleep of her own or being helped by tab. diazepam 5mg or lorazepam 1 or 2 mg at bed time. In 10% primigravidae developing preeclampsia by 28-32 weeks, BP rises from basal level of 110/120/60-80 mmHg following above regime. Thereafter she is instructed strictly to follow the regime. She suspends all indoor and outdoor activities. Fortnightly clinic checkup shows halting of BP at 140/90 - 150/100 mmHg. Some of them develop generalised oedema for which she avoids excess water drinking and extra salt and day time rest on lateral lying 2-3 hours a day. On this her oedema lessens on subsequent checkups.

Checkups. For normal growing low risk pregnancy, antenatal woman gets five checkups on 10th, 18th, 24th, 32nd and 36th week. Preeclampsia developing woman gets 10 checkups or more finally admitting her into antenatal ward for rest. As soon as she develops hypertension at 140/90 mmHg at clinic visit, she gets fortnightly clinic B.P. checkups, bringing her weekly BP checkup record by the family physician. Careful BP checkup (preferably by the same observer) is key care in preeclampsia. At every clinic checkup her urine is tested for proteinuria.

Ninety percent women under above regime can keep her BP halted at mild preeclampsia (Stage I), 10% who failed to follow the regime or can not sleep even on diazepam or lorazepam develops stage II disease.

Other Drugs. No directic, no antihypertensive drug is required for stage I disease.

Hospital admission : (1.) Stage I disease continues clinic care till 38 weeks when she is admitted (2) stage I disease developing proteinuria and stage II disease are admitted into the hospital.

Author's experience. During last 10 years 1050 primigravid women are cared privately by the author on Rule of Ten. On an average 10 ± 1.90 (9-14) kg maternal weight gain is obtained on food and rest.

One hundred fifteen patients (10.9%) developed stage I preeclampsia and II (1.4%) developed stage II disease.

Delivery. In stage I all came to spontaneous labour by 40th week. Labour induction was done in case 40 weeks was certain'y crossed.

Birth weight. Mild preeclampsia under care as above is a benign disease. It does not cause IUGR when maternal weight gain was around 10 kg.

Acknowledgement

The author is clinical researcher on preeclampsia since 1955. From 1955-1960 he worked for Ph.D thesis on the role of dietetic deficiency in causation of preeclampsia. No statistical significant correlation could be found between any dietetic deficiency and preeclampsia.

References

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